THE VETERINARY JOURNAL

Editors :

T. DALLING, M.A., M.R.C.V.S.

J. McCUNN, M.R.C.V.S., M.R.C.S., L.R.C.P.

NOVEMBER, 1944

EDITORIAL

TUBERCULOSIS

We publish in this issue an article which no doubt will promote much comment, for it deals with an old enemy in a way that is not usual, but which has not lacked the support of a minority since the time of Pasteur and Koch. The author has been at some pains to search for evidence which substantiate his claims, and now that he has set them down in print the reader can assess their value, true or false. If the ideas so annunciated can be supported after a critical analysis then present-day conceptions of the prophylaxis and treatment of tuberculosis should undergo a revolutionary change. Tuberculosis in its various forms has ravaged both man and beast from time immemorial. How it originated we do not know, but history tells us, and there is evidence in support, that it was prevalent in ancient Egypt thirteen centuries and in India fifteen centuries B.C. Hippocrates encountered it in Greece in the fifth century and Aretæus in Asia Minor in the first century B.C. These records primarily concern its occurrence in man, but we also know that its presence in animals was not unnoticed, for the Mosaic Law prohibited the use for food of the flesh of animals affected with the disease. And so it has continued down the ages. The Talmud of the first, the Mishna of the third and the Gemara of the fifth centuries A.D. similarly prohibit the consumption of tuberculous flesh. In more recent times we note that it was recognised to be a contagious disease by Rubling (Germany) and Huzard (France) in the latter part of the 18th century A.D. Villemin in 1865 was probably the first to place on record the fact that it could be contracted from an animal of another species. Robert Koch in 1882 provided the greatest landmark in the story of tuberculosis by his discovery of the causal organism and many thought that the battle was almost

XUM

over, but it was only the first page in the story. Rivolta (1889) and Maffucci (1890) demonstrated that the avian organism differed somewhat from the mammalian germ, and Theobald Smith proved that bovine and human tuberculosis were not caused by identical organisms, although he stated that they were closely allied.

Then came the memorable battle between MacFadyean and Koch. MacFadyean contested Koch's statement that bovine tuberculosis was not infective to man. A subsequent Royal Commission proved MacFadyean to be correct, and this decision has influenced all our efforts since that date. Steps had to be taken to allay the fears of an anxious public. Regulations were devised and enforced to prevent the dangers now known to exist. These regulations varied in their intensity according to the countries in which they were promulgated. What has been the result? Are we satisfied with the progress made? There can be no doubt that they have done much to promote a more wholesome meat and milk supply, but it cannot be denied that tuberculosis still exists even in those countries in which regulations were the most rigorous. Where there has been a decrease in human tuberculosis many are of the opinion that the decrease is not due to any regulations devised for tuberculosis itself. Many other factors have been at work, such as a lowering of the virulence of the organism, a step up in the powers of resistance of the potential victims, and better housing, sanitation and nutrition. We must also remember that all infective diseases conform to a common law of rise and fall which is regulated by variations in the resistance of the patients and of the virulence of the infecting organism. If one makes a chart of the incidence of any particular infective disease over a period of time it will be marked by a more or less regular series of rise and fall which can be correlated with these variations in resistance and virulence. Good examples of such rise and fall can be quoted as occurring within a comparatively short period of time, i.e., scarlet fever and measles. Within a period of 50 years these two diseases have almost changed places. Scarlet fever has become less dangerous, whereas measles has increased in its lethal effects.

The distance between the curves when we chart the incidence of tuberculosis may be greater, indicating a longer period of time, but the basic natural laws ruling an infective disease are the same. The school of thought to which Mr. Rainey evidently belongs also believes that there are considerable variations in virulence of an organism such as that of tuberculosis depending upon whether the germ is the specific one for the species involved or if it is derived from an animal of another species. They contend that the bovine organism is not as dangerous as it is generally supposed to be to humans. Some go further and assert that it may be beneficial for being heterogeneal it is of low virulence, will not produce the disease in a serious form and will stimulate resistance after the manner of an attenuated live vaccine.

There is no doubt that the most lethal organism for humans is the mycobacterium tuberculosis (hominis). Also there is no doubt that the M.T. (bovis) is responsible for a relatively small number of cases of tuberculosis in man. M.T. (bovis) would appear to confine its activities in man to lymph nodes, bowels and joints, and it is a recognised fact that the subject recovered of such an infection rarely contracts the disease as engendered by the M.T. (hominis).

XUM

General Articles

BLOOD PICTURE OF A COW DURING A NORMAL PREGNANCY AND PARTURITION

By P. G. D. MORRIS, M.A., M.R.C.V.S.,
Histology Department, Royal (Dick) Veterinary College, Edinburgh

Introduction

It was decided that an examination of blood samples taken at weekly intervals from a pregnant cow throughout the period of pregnancy might reveal some interesting changes in the blood picture.

The cow chosen for the purpose was a Shorthorn belonging to the College.

Throughout the whole period of pregnancy she was kept in a stall, stall fed, and never put out to grass during that time.

Historical

R.B.C. and Hæmoglobin

According to Burnett,[1] the effect of pregnancy and parturition in the different species of animals has not yet been determined in a sufficient number of cases to warrant making any definite statement for each.

He says that normal pregnancy does not appear to affect the number of red corpuscles as a rule, though Cohnstein[2] found an average of 9,742,000 R.B.C.s and 7.8 per cent. hæmoglobin in seven pregnant sheep and an average of 12,090,000 corpuscles and 5.5 per cent. hæmoglobin in five non-pregnant sheep.

Parturition, Burnett says, seems to lower the R.B.C. count for a short time.

Burnett and Traum[3] found that the R.B.C. count remained low in a bitch for two or three weeks after parturition.

According to Raphael Isaacs,[4] many otherwise healthy women may show a slight decrease in the number of R.B.C.s during pregnancy, but he says it is probable that there is no constant deviation from the normal.

Bland, Goldstein and First[5] found a red blood cell count of less than 3.5 millions per cmm. in 42 per cent. of three hundred maternity patients and no correlation with parity, age, blood pressure or social conditions was evident.

A rapid return to normal was noted after delivery.

Numerous authors, including Spiegelberg and Gescheiden,[6] Wild,[7] Adachin,[8] Terhola,[9] Von Limbeck[10] and Engelsen,[11] found little or no change in the R.B.C. count or hæmoglobin content during pregnancy.

R. Schroeder,[12] Carton[13] and Zangemeister[14] reported an increase, whereas Fouassier,[15] Kosina and Ekert,[16] P. J. Meyer,[17] Dubner,[18] Henderson,[19] Bar Daunay,[20] Blumenthal[21] and H. A. Dietriel[22] noted a decrease in the number of erythrocytes.

Others, Ingerslev,[23] Thompson,[24] Owen (1906), Sondern[25] and Gram,[26] describe increases and decreases in the R.B.C. count during various stages of pregnancy.

Strauss[27] attributes the so-called physiological anæmia of pregnancy to approximately 20 per cent. increase in blood volume during the last trimester.

XUM

Leucocytes

Burnett and Traum found a leucocytosis in a bitch reaching 23,000 at the time of parturition, then dropping to normal in less than three weeks.

Storch[28] found no increase in leucocytes in pregnant cows or goats, and only a slight increase in a small percentage of pregnant sheep examined.

According to Burnett, during the later period of pregnancy in woman there is usually a moderate increase in the number of leucocytes. It is most marked in the primiparæ; in multiparæ it occurs in about 50 per cent. of the cases. It is a mixed leucocytosis, the percentages remaining unchanged except that the eosinophils may not be increased. After parturition the leucocytes gradually decrease, reaching normal in about 4-14 days.

Examination of Blood Samples

Technique

The blood was taken directly from the jugular vein by means of a hypodermic needle. Samples were taken at weekly intervals except after parturition, when samples were taken 36 hours, 8½ days, 15 days, 3 weeks and 5 weeks after calving.

R.B.C. and W.B.C. counts were made using the Metz hæmocytometer.

Hæmoglobin per cent. was estimated by means of a Dare hæmoglobinometer (100 per cent. = 13.77 grms. in 100 c.cs. of blood) and a Sicca hæmoglobinometer (100 per cent. = 13.85 grms. per 100 c.cs. of blood), the average of the readings from the two instruments was the figure recorded.

Colour Index was calculated by means of the formula:

$$\text{Colour Index} = \frac{\%\ \text{Hæmoglobin}}{\text{number of R.B.C.s expressed in millions} \times 20.}$$

Differential Counts.—At least three blood films were made from each sample and stained by Piney Pappenhein's modified method. A hundred W.B.C.s were counted on each film, making a total of three hundred W.B.C.s counted for each sample.

The figures recorded were the average of the three counts.

Findings

The Red Blood Cells (see Graph I)

The R.B.C.s showed a tendency to fluctuate throughout the period of pregnancy. In the early stages there was a slight increase in the number. This increase reached a peak in the 8th week (7,540,000 per cmm., the highest count recorded during the examination), followed by a steady decrease till the 17th week. The count was then 5,680,000 per cmm., the lowest count recorded.

The count during the 18th week showed a sudden rise to 7,160,000 per cmm.; this was followed by a decrease in number till the 20th week.

During the period 20th to 23rd week, there was an increase in the count. After the 23rd week a gradual decrease (from 7,306,000 to 6,100,000 per cmm.) in the count was noted until parturition.

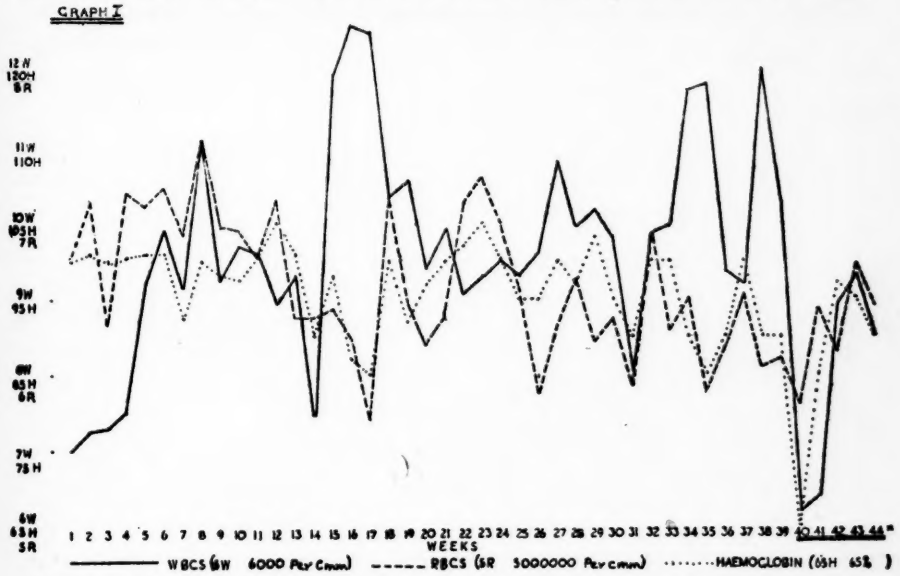
Thirty-six hours after calving the R.B.C. count was 5,790,000 per cmm.

Eight and a half days after calving the count was 6,420,000 per cmm., 15

days after calving 6,150,000 per cmm., 21 days after calving 6,715,000 per cmm., 5 weeks after calving 6,450,000. (So from the above figures it appears that there is a slight decrease in R.B.C.s during pregnancy, especially in the later stages, followed by a rapid return to normal after parturition.)

Hæmoglobin (see Graph I)

There was only a slight fluctuation in hæmoglobin percentage throughout pregnancy. The fluctuations of the hæmoglobin followed the rise and fall in R.B.C.s except during the 28th week and 29th week; here the R.B.C.s showed



GRAPH I.—Figures 40, 41, 42, 43, 44 (underlined). 40 = 36 hours after calving. 41 = 8½ days after calving. 42 = 15 days after calving. 43 = 3 weeks after calving. 44 = 5 weeks after calving.

an increase in the 28th week, the hæmoglobin a decrease, while in the 29th week the R.B.C.s decreased and the hæmoglobin per cent. showed an increase.

The highest percentage hæmoglobin recorded was 105 per cent. in the 12th week, and the lowest 65 per cent. 36 hours after calving.

The Colour Index

The average for the 44 weeks during which blood samples were taken was .73.

The highest figure recorded for the colour index was .82, during the 29th week. The lowest, .56, was recorded 36 hours after calving; 8½ days after calving the colour index was .66; 15 days after parturition the colour index returned to normal (.79).

W.B.C.s (Total) (see Graph I)

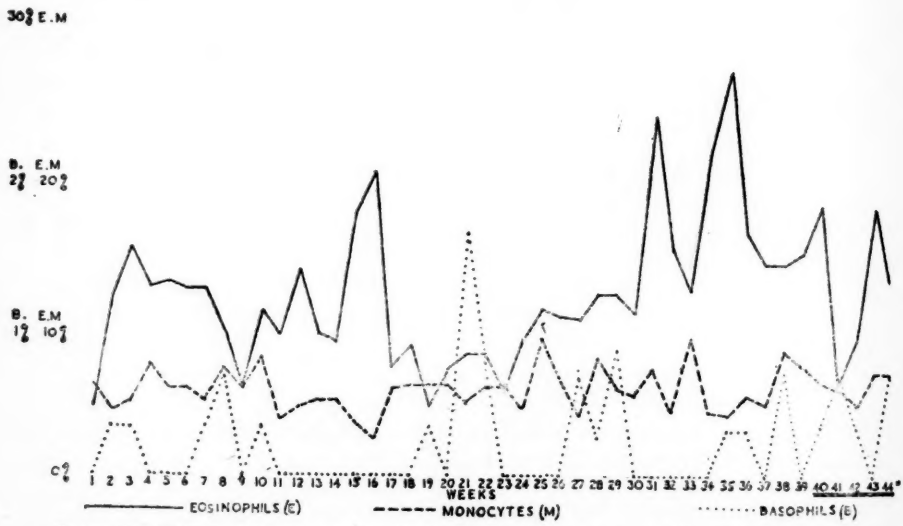
The number of W.B.C.s gradually increased during the early weeks of pregnancy, reaching a peak (11,066) at the 8th week; the numbers then decreased

XUM

to 7,400 in the 14th week: during the 15th week there was a sudden increase in the number of W.B.C.s (11,933); that persisted for approximately five weeks until the 20th week, when the count fell to 9,350. Their numbers then remained fairly constant until the 31st week, when a sudden decrease was recorded, followed by a well-marked rise, covering the period from the 32nd to the 35th week, succeeded by a return to the average for the weeks 36th to 37th.

A definite increase was recorded over the period 38th to 39th weeks. Thirty-six hours after parturition a very sudden decrease was recorded (6,200); 8½ days after, the count was still low (6,400); 15 days after, 8.400, indicating a return to normal that persisted from the 15th to 19th weeks.

GRAPH II.



GRAPH II.—Figures 40, 41, 42, 43, 44 (underlined). 40 = 36 hours after calving. 41 = 8½ days after calving. 42 = 15 days after calving. 43 = 3 weeks after calving. 44 = 5 weeks after calving.

The sudden increase in W.B.C.s from the 15th to 19th weeks coincided with final cessation of lactation.

The leucocytosis recorded during the 38th to 39th weeks (the last two weeks before parturition) agrees with the findings of Burnett, Traum and Storch.

Neutrophils (see Graph III)

The average number of neutrophils was 2,721.51 per cmm. of blood throughout the whole period over which the blood samples were taken.

During the period 15th, 16th and 17th weeks, which coincided with the cow "drying off," quite a definite increase in the number of neutrophils was noted; the highest figure recorded during those weeks being 3,356.2 per cmm.

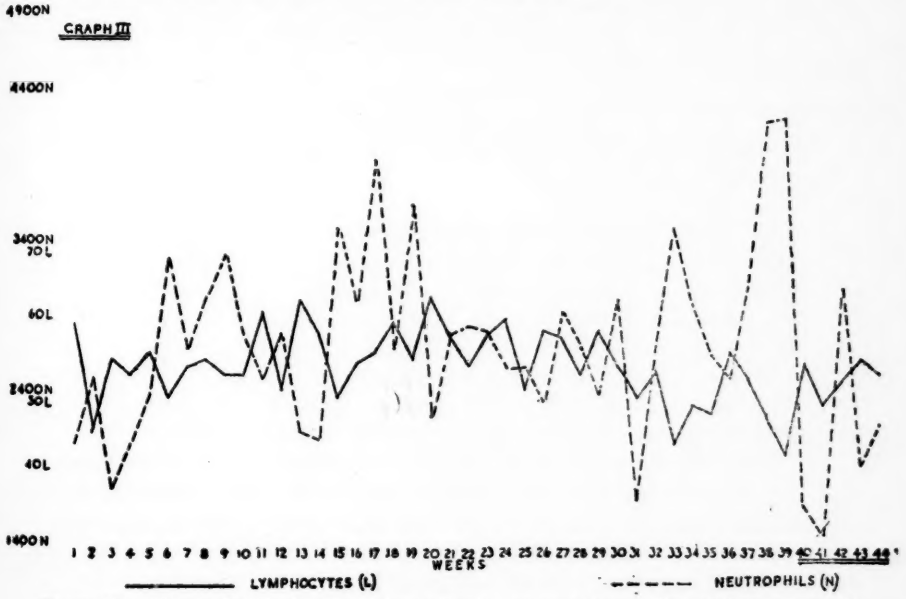
From the 17th week to the 31st week the number of neutrophils showed only a slight fluctuation; in the 31st week, however, there was a distinct decrease to 1,679 per cmm., followed by an increase to 3,514 per cmm. in the 33rd week.

XUM

The sudden rise in the 33rd week was probably due to a skin condition from which the animal was suffering during the period (31st to 33rd week). The decrease in the 31st week coincided with the commencement of the skin condition.

After the 33rd week the neutrophils decreased till the 36th week. Over the period 37th week, 38th week and 39th week, the neutrophils increased in numbers, reaching a peak figure of 4,192 per cmm. This was the highest number recorded during the period of pregnancy.

The count made 36 hours after calving showed a sudden fall in neutrophils to 1,652 per cmm.; 8½ days after calving an even lower figure was recorded, 1,473 per cmm.; 15 days after calving the neutrophils returned to normal (average for the 42nd, 43rd and 44th weeks being 2,449 per cmm.).



GRAPH III.—Figures 40, 41, 42, 43, 44 (underlined). 40 = 36 hours after calving. 41 = 8½ days after calving. 42 = 15 days after calving. 43 = 3 weeks after calving. 44 = 5 weeks after calving.

The highest proportion of the neutrophils were found to be Poly I (19.8 per cent. of total W.B.C.s).

Only on two occasions were myelocytes or metamyelocytes observed (.33 per cent. 6th week and .33 per cent. 40th week).

Band Forms were seen on 17 occasions; the highest percentage (3.66 per cent.) was recorded during the 40th week (36 hours after calving).

Poly II.—The average percentage for this type of cell was 7.65 per cent. of the differential count.

The highest percentage recorded was 13 per cent. recorded during the 39th week (the week prior to calving).

The lowest percentage recorded was during the 8th week (3.66 per cent.), in spite of the fact that during this week there was an increase in neutrophils.

XUM

Poly III.—The average figure recorded was 1.43 per cent.

Highest percentages observed—

8th week	...	2.66%
*33rd week	...	3.66%
39th week	...	3.00%

Lowest percentages recorded—

3rd week	...	.33%
14th week	...	.33%
40th week	...	Nil

* Probably reaction to skin condition.

Poly IV.—This class of neutrophil was only recorded twice throughout the period of blood examination.

During the 19th week, .166 per cent., and .25 per cent. in the 43rd week (three weeks after calving).

Eosinophils (see Graph II)

The average over the whole period of blood examination was 11.77 per cent.

Highest percentages recorded—

3rd week	...	16.00%	
15th week	...	17.00%	(" dried off ")
16th week	...	20.00%	(" dried off ")
*31st week	...	23.66%	(skin condition bad)
*32nd week	...	15.00%	(skin condition improved)
*33rd week	...	12.33%	(skin condition improved)
*34th week	...	21.33%	(skin condition worse)
*35th week	...	26.66%	(skin condition bad)
*36th week	...	12.00%	(general clearing up skin condition)

* During these weeks the animal was suffering from a non-parasitic and non-infectious skin condition, possibly due to a vitamin deficiency.

The decrease in eosinophils in the 33rd week appears to coincide with an improvement of the skin condition. During the 34th and 35th weeks the skin condition flared up again, finally cleared up again in the 36th week.

There was an increase in eosinophils to 17.75 per cent. during the 43rd week (three weeks after calving).

No increase in eosinophils was observed immediately prior to parturition.

Lowest percentages recorded—

1st week	...	4.33%
19th week	...	4.66%
41st week	...	6.33%

Basophils (see Graph II)

Average .22 per cent. over the whole period of 44 weeks.

On 18 occasions basophils were recorded in the differential counts.

The highest percentage recorded was 1.66 per cent. during the 21st week.

It was thought that perhaps some correlation might be found between the appearance of basophils and the eosinophils count, but this does not appear to be so, although they did appear a few weeks before increases in the eosinophils.

XUM

The 1.66 per cent. recorded was not, however, followed by any increase in eosinophils; not, anyway, until weeks later.

Lymphocytes (see Graph III)

53.57 per cent. was the average recorded over the whole period of the blood examination.

The variation in the percentage of lymphocytes recorded week by week was very small.

Highest percentages recorded—

11th week	...	61.00%
13th week	...	62.66%
20th week	...	63.00%
24th week	...	60.00%

Lowest percentages recorded—

2nd week	...	45.66%	(sudden increase of neutrophils)
33rd week	...	43.33%	(during skin condition)
39th week	...	42.00%	(rise in neutrophils week prior to parturition)

Monocytes (see Graph II)

Average was 5.65 per cent. for the period of blood examination.

Highest percentages recorded—

4th week	...	7.33%
8th week	...	7.00%
10th week	...	7.66%
25th week	...	9.00%
28th week	...	7.75%
33rd week	...	9.00%
38th week	...	8.33%
39th week	...	7.25%

Lowest percentages recorded—

15th week	...	3.50%	(cow going "dry")
16th week	...	2.33%	(cow going "dry")
24th week	...	4.00%	

Summary

Since only one animal was examined it is impossible to make any definite statement of changes in the blood picture during pregnancy for bovines in general, but the findings do substantiate those of some other workers.

The R.B.C.s showed a slight decrease in the latter half of the gestation, a marked decrease was noted 36 hours after parturition with a rapid return to the normal.

Hæmoglobin showed slight fluctuation, with a sudden fall 36 hours after parturition, followed by a return to normal in about 15 days.

A slight leucocytosis was noticed at the time the cow was finally "drying off" from the previous lactation: a second leucocytosis was noted during the two weeks immediately prior to parturition.

The leucocytosis in both cases was mainly due to an increase in neutrophils, but during the leucocytosis noted when the cow was "drying off" there was a

XUM

definite increase in eosinophils, while during the leucocytosis immediately prior to parturition there was no increase in eosinophils; in fact, there was a decrease.

Lymphocytes remained fairly constant in numbers throughout the period of blood examinations.

Eosinophils showed a marked increase in the final stages of lactation and during the weeks the animal was suffering from the skin condition.

Monocytes were more or less constant in number, but showed a definite decrease for the 15th and 16th weeks, those weeks coinciding with the "drying off" from the previous lactation.

Appreciation

My thanks are due to Professor T. Grahame for his helpful suggestions and guidance, to the Clinical Department for their help in the taking of the blood samples, and to Mr. William Neill for his help with the counts.

REFERENCES

(1) Burnett, S. H. (1917): "Clinical Pathology of the Blood of Domesticated Animals," p. 63.
(2) Cohnstein, J. (1884): "Blutveränderung während der Schwangerschaft." *Arch. f. gesamm. Physiol., 34,* 233.
(3) Burnett and Traum (1905): "The Clinical Examination of the Blood of the Dog." *Proceed. Am. Vet. Med. Ass., 13,* 349.
(4) Raphael, Isaacs: *Downey's Handbook of Hæmatology,* Vol. 1, p. 55.
(5) Bland, P. B., Goldstein, L., and First, A. (1930): "Secondary Anæmia in Pregnancy and in Puerperium. A Study of 300 Cases." *Am. J. M. Sc., 179,* 48.
(6) Spiegelberg, O., and Gescheidlen, R. (1872): "Undersuchungen über die Blutmenge trächtiger Hunde." *Arch. f. Gynäk., 4,* 112.
(7) Wild, M. (1897): "Untersuchungen über den Hæmoglobingehalt und die Anzahl der rothen and weissen Blutkorperchen bei Schwangeren und Wochrer innen." *Arch. f. Gynäk., 53,* 363.
(8) Adachi, S. (1912): "Zur Frage der Blutveraenderung bei Schwangeren und Gebaerenden." *Beitr. z. Geburtsh u. Gynäk., 17,* 174.
(9) Terhola, L. (1914): "Ueber Blutveränderungen während der Geburt, Laktationsperiode und der ersten Menses postpartum." *Arch. f. Gynäk., 103,* 115.
(10) Von Limbeck, R. R. (1896): *Grundriss einer Klinischen Pathologie des Blutes.* Jena.
(11) Engelsen, E. (1884): "Undersoegelser over blodlegemernes Antae, Hæmoglobinmaengle og Stoerrelse." *Disputats.* Copenhagen: W. Prior.
(12) Schroeder, R. (1891): "Untersuchungen über die Beschaffenheit des Blutes von Schwangeren und Wöchnerinnen, sowie über die Zusammensetzung des Fruchtassers und ihre gegenseitigen Beziehungen." *Arch. f. Gynäk., 39,* 306.
(13) Carton (1943): "Modifications du sang pendant l'accouchement et les suites de couches normales et pathologiques." *Ann. d. gyn. et d'obst., 30,* 161.
(14) Zangemeister, W. (1903): "Die Beschaffenheit des Blutes in der Schwangerschaft und der Geburt." *Ztschr. f. Geburtsh u. Gynäk., 49,* 92.
(15) Fouassier, J. J. A. (1878): "De la numération des globules du sang dans les suites de couche physiologique et dans la lymphangite utérine." *Abst. Rev. mens. des méd. et de chir., 2,* 559.
(16) Kosina and Ekert (1883): "Untersuchungen des Blutes Gebaerenden und Woechnerinner Nach dem Verfahren von Hayem und Nachet." *Med. Westnik.*
(17) Meyer, P. J. (1887): "Untersuchungen über die Veränderungen des Blutes in der Schwangerschaft." *Arch. f. Gynäk., 31,* 145.
(18) Dubner (1890): "Untersuchungen über den Hæmoglobingehalt des Blutes in den letzen Monaten der Graviditaet und im Wochenbette." *München. med. Wchnschr, 37,* 517; 537; 551.
(19) Henderson, J. (1902): "Observations on the maternal blood at the term and during puerperium." *J. Obst. and Gynec.,* Brit. Emp. 1, 168.
(20) Bar and Daunay (1904): "Proportion du plasma, richesse en globules et en hémoglobine; Alcalinité du sang à la fin de la grossesse normale." *Compt. rend. Soc. de biol., 56,* 105.
(21) Blumenthal, R. (1907): "Ergebnisse der Blutuntersuchungen in der Geburtshülfe und Gynækologie." *Beitr. z. Geburtsh. u. Gynäk., 11,* 414.
(22) Dietrich, H. A. (1911): "Studien ueber Blutveraenderungen bei Schwangeren, Gebaerenden and Woechnerinnen." *Arch. f. Gynäk., 94,* 383.

XUM

(23) Ingerslev, E. (1897): "Ueber die Menge der rothen Blutkörperchen bei Schwangeren." *Centralbl. f. Gynäk., 3,* 635.
(24) Thompson, W. L. (1904): "The blood in pregnancy." *Johns Hopkins Hosp. Bull., 15,* 205.
(25) Sondern, F. E. (1906): "Anæmia in pregnancy." *Bull. Lying-in Hosp., N.Y., 3, 27.*
(26) Gram, H. C. (1920): "Physiological anæmia of pregnancy." *Ugeskr. f. Laeger,* Copenhagen, *82,* 1609.
(27) Strauss, M. B. (1934): "The etiology and treatment of anæmia in pregnancy." *J.A.M.A., 102,* 281.
(28) Storch, A. (1901): "Untersuchungen über den Blutkorperchengebalt des Blutes der landwirtschaften Haussangeriere." *Inaug. Diss. Bern, 1901.*

THE RELATIONSHIP OF BOVINE TO HUMAN TUBERCULOSIS CONSIDERED IN THE LIGHT OF EXPERIENCE

By J. W. RAINEY, C.B.E., M.R.C.V.S.,
Tasmania

OBVIOUSLY so complex a study as the relationship of bovine to human tuberculosis in all its aspects could not be fully discussed in a brief article, even if the writer were competent to do so, which he is not.

What follows then is a rather dillettant effort to show up the highlights of the question as they have been revealed to a sceptical and inquisitive mind observing and reflecting upon relevant facts during a long time and in many places.

Robert Koch and John MacFadyean

The writer's curiosity in the matter was first aroused by the perplexing divergence of view shown in the classical London debate between Robert Koch and John MacFadyean more than forty years ago.

Koch, it will be recalled, strongly denied, and MacFadyean as vehemently affirmed, the infective danger of bovine tuberculosis bacillus for the human being, and MacFadyean was judged to have won the day. Having regard to the calibre of both men it was always, in the present writer's opinion, inconceivable that two such absolutely opposite views could have been drawn from identical experience. British experience on the matter, one felt, must to some extent have differed from Continental experience.

Impressions formed from experience are sometimes hard to support by exact data, but they may still be fairly correct, and it may be that Koch was led by such experience to his conclusion without being able to defend his view and without realising that it had only a local validity.

The same applies to MacFadyean, who told the truth, say, for Scotland, but not, as will be shown later, for, say, Tasmania, Queensland or S. Africa.

Devon and Cornwall Tried Out Milk Sterilisation Before Pasteur

Another aspect of the question which early intrigued the writer (when pasteurisation as a means of safeguarding the human being from the cow first became the rage) was that of the relative incidence of tuberculosis in general and non-pulmonary tuberculosis in particular affecting the human being in

XUM

Devon and Cornwall, as compared with the rest of England, at a time when unsterilised milk or milk products, only excepting junket, were almost unknown in rural areas west of Exeter.

The heat-sterilisation of milk had been in vogue in sunny rural Devon and Cornwall from time immemorial, yet there was probably no part of England where tuberculosis in all its forms took a heavier toll of life in the nineteenth century. That is comparing rural areas with rural areas: it would not be fair to compare rural areas with industrial areas.

Why was this, and why is this significant and immense experiment in the prophylactic value of heat-sterilisation never mentioned when and where pro-pasteurisers foregather?

Experience in Fiji

It was in the Fiji Islands in 1919-20 that the writer first studied in a practical way the variability of infectivity of the bovine tuberculosis bacillus as it occurred under natural conditions among animals living wholly in the open.

In order to make a rough estimate of the incidence of tuberculosis among cattle in Fiji, about 30,000 animals were examined individually, some scores of open or suspected clinical cases were shot and dissected, a careful record was kept of tuberculosis incidence over a period in the main abattoirs, and a good many tuberculin tests were undertaken. The greater part of the Fijis is in a wet belt, but there is a sharply defined dry belt in the main island (known as Viti Levu), where the rainfall is less than half of the wet belt. The division is so sharp that there is no shading off of the forest into savannah: the trees stop suddenly and one debouches from the jungle on to a relatively dry plain.

It was found that, whereas the incidence of tuberculosis among cattle in the dry belt was about 2 per cent., with no generalised cases, the incidence in the wet belt was around 20 per cent. with some, but not many, generalised cases.

There was no sort of fence between the dry and the wet belt, and cattle were occasionally passing from one belt to the other, limited in this movement only by the difficulty of traversing the forest of the rain belt. Consequently the factors limiting the incidence of tuberculosis in the dry belt must have been always at work, checking the spread of the disease and reducing the virulence of the bacillus.

An Essential Question

At once then the question arose and demanded a reply—as it still arises and demands a reply wherever such variations in the infectivity are found. That is, "What is the corresponding variation of difference in the infectivity of the bovine bacillus for the human being?"

If one finds, as one found in Fiji in 1920 and finds in Tasmania in 1944, areas where, owing to certain physico-chemical factors, the bovine tuberculosis bacillus can survive only rarely, and with difficulty, in its natural host—the cow—what is the danger in such areas of human infection by such a languid germ trying in vain to get a foothold on an exotic host?

The answer—the writer believes—is practically nil: there is practically no danger for the human being in such circumstances.

One cannot say ever of any potentially infective germ that there is abso-

XUM

lutely no danger, just as one cannot say for certain that walking down stairs or a hearty meal will never be dangerous.

But one can affirm, both from a common-sense point of view and on the basis of abundant statistical evidence, that the danger of tuberculous infection of bovine origin for human beings is practically non-existent in areas where the infectivity of the bovine bacillus for its natural host is obviously very low.

There is—the writer thinks—a formula, capable of being found statistically, whereby it would be possible to state definitely that when the incidence of the bovine disease naturally falls below a certain point the danger to the human being virtually disappears.

The writer would go even further than this. He would say that below such a point the existence of bovine tuberculosis in its natural host is more beneficial than harmful for the human being. It may help to confer immunity. At this point the reader will become restive and demand some proof, and this proof will now be offered in the form of Australian experience in general and Tasmanian experience in particular.

Australian Experience

An invaluable basis for the study of Australian experience in this connection is available in an outstanding paper, entitled " Tuberculosis in Australia," by M. J. Holmes, M.B., Senior Medical Officer, Commonwealth Department of Health, Canberra, read at the Australian Medical Congress in 1937 and published in the *Medical Journal of Australia* for November 6, 1937.

Full statistics, dating back to 1870, for all forms of human tuberculosis in each state are given.

The statistics for Queensland are especially interesting to the veterinarian, because, as will be shown hereafter, they prove beyond doubt that a rapid and unbroken decline in non-pulmonary human tuberculosis can take place without the heat-sterilisation of milk, without eradication of tuberculous cows, and, indeed, without our help at all.

In Queensland, non-pulmonary tuberculosis in human beings fell from an annual death rate of 63.4 per 100,000 of population in the period 1885-1889 to 2.3 per 100,000 in the period 1935-1936. Thus the present annual rate of mortality is less than 3 per cent. of the rate 40 years ago, constituting a greater and more dramatic decline than the simultaneous decline in the United States without any equivalent national effort having been made to achieve that end.

Queensland, so far as human health is concerned, has had for nothing, and without troubling anyone, what it has cost the United States hundreds of millions of dollars and endless worry to stock-owners to accomplish.

It is true that since 1910, or thereabouts, there has been an increasing veterinary attempt at eradicating tuberculous cows in Queensland, and quite lately there has been some fuss about pasteurisation, but these measures in so vast an area as Queensland did not amount to much and certainly have had no significant effect on the vital statistics of human tuberculosis.

For instance—and this is the all-important point—a very much greater decline in human mortality from non-pulmonary tuberculosis occurred prior to 1910 than has occurred since. From 1889 to 1909, without veterinary control, and without any pasteurisation, the annual mortality from non-pulmonary human

XUM

tuberculosis, in round figures, fell fifty points—from 63 to 10. Since 1909 the annual mortality has fallen only eight points, from ten to two. Logically, therefore, it seems incontestable that in Queensland the eradication of tuberculous cows has had no advantageous effect on the decline in human tuberculosis.

During the same periods the decline in pulmonary tuberculosis in man, which nowhere is attributed to infection from bovine sources, has been from an annual death rate of 147 per 100,000 of population to 34.6.

That the present death-rate from pulmonary tuberculosis, into which the bovine factor does not enter, is 27 per cent. of the 1889 rate, constituting a drop only to about one-fifth of the 1889 rate, as against a decline to one-thirtieth in the instance of non-pulmonary tuberculosis, with the bovine factor given a free field for most of the time.

Experience in Tasmania

Dr. Holmes, in his paper, observes that the statistics of tuberculosis in Tasmania are strikingly different from those of Queensland, and comments that "The determination of the reasons for the different behaviour in Tasmania and in Queensland should prove a fruitful field for investigation."

The present writer has had the honour to carry out this investigation, which has—as Dr. Holmes foretold—proved fruitful indeed, though not perhaps in the direction imagined by Dr. Holmes. Statistics show that the decline in mortality from non-pulmonary tuberculosis in human beings in Tasmania has been from an annual peak of 23.4 per 100,000 in 1885-1889 to 9.2 in 1933-34.

Simultaneously the decline in the annual mortality from respiratory tuberculosis in Tasmania has been from a peak of 105-8 to 47 per 100,000.

This extraordinary difference from the Queensland figures seems to have appeared to Dr. Holmes as indicating infection from bovine sources, and it was this point that the writer set himself to investigate on assuming veterinary duty in Tasmania in 1940. The following main questions presented themselves: Was bovine tuberculosis prevalent in Tasmania and was there any medical proof of human infection from bovine sources? The answer to both questions—as will be shown—is in the negative. There is actually less bovine tuberculosis on the whole in Tasmania than in Queensland, and, indeed, than in any other British country known to the writer.

In his own district—an intensive dairying area of about 400 square miles on the North-West Coast of Tasmania, with a sunshine ratio of about 2,300 hours a year—he has not found in more than three years of active clinical work a single case of bovine tuberculosis among animals bred in the district. During the same period, out of about 8,000 bovine animals, largely cows, slaughtered in the local abattoirs, only three indisputable generalised cases of tuberculosis have been confirmed by autopsy, and these animals came from another part of the island.

Yet, in the writer's district, the incidence of human tuberculosis in general is rather higher than in some other parts of Tasmania, and there is no known difference in the incidence of non-pulmonary tuberculosis.

As regards tuberculin testing, out of 412 cows tested to date by the writer

there was only one positive reaction. Autopsy failed to reveal the slightest naked-eye evidence of tuberculosis.

During the same period, a neighbouring colleague, Mr. H. O. Cartwright, B.V.Sc., tested 2,475 cows, with 14 reactors, or an incidence of about .5 per cent.

The municipal veterinary officer for Launceston, Mr. D. Roche, B.V.Sc., has informed the writer that, during the same period, he has tested about 1,200 cows, with only two reactors.

In another part of the North-West Coast, where the rainfall is fairly heavy, 3,396 cows were tested, with 3.8 per cent. of reactors, but the District Veterinary Officer, Mr. A. F. Ryan, B.V.Sc., has informed the writer that the autopsies for the most part revealed little or no naked-eye evidence of disease.

What are termed euphemistically " non-visible lesions " prevailed. The writer regards this most unscientific term as a euphemism used to excuse the wasteful destruction of animals whose natural resistance would have been passed on as inherited quality.

In view of the above facts it may, then, be stated definitely that bovine tuberculosis in a form that could possibly be infective for man is extremely rare and may be non-existent in Tasmania. This fact is reflected in the typing of the tubercle bacilli found in human cases of disease treated at Hobart.

Dr. Duncan, Government Pathologist at Hobart, from his experience in such typing, insists that there is no bovine infection of human beings in Tasmania.

If there is any truth at all in the theory—put forward by others and accepted by this writer as reasonable—that human ingestion of the bovine tubercle bacillus does to some extent confer immunity on the human being, it is in Tasmania rather than any other part of Australia that we should expect to find the highest incidence of human tuberculosis in general and the non-pulmonary form in particular. And that is exactly what we do find, and here is a possible part of the answer to the problem posed by Dr. Holmes. Not by any means the whole answer, or even the most of it, but a possible and even a probable part of it.

In Queensland, for instance, where the incidence of human tuberculosis is so much less than in Tasmania, recent tests by Government veterinary officers showed that out of 594 cows tested for tuberculosis, and subsequently examined post-mortem, 11 per cent. showed lesions; and the writer is informed that this incidence is fairly representative of cows in the coastal districts of Queensland.

Why is the Incidence of Bovine Tuberculosis Rare in Tasmania?

This is the key question in this paper and it applies not only to Tasmania but to many other parts of Australia, as well as to South Africa, perhaps to the Channel Islands, and even to parts of Devon and Cornwall.

The writer affirms that the answer is simple, easy, and definite. It is that the bovine bacillus loses more or less its pathogenicity wherever there can be and is consistently close cropping of herbage with a sunshine ratio exceeding 2,000 hours a year and cows are kept in the open air all the year round.

The last-stated condition is probably the least essential of the three and may not be essential at all.

XUM

Obviously it is not enough that cattle should live wholly in the open, because the cows in the wet belt of the Fiji Islands, as well as the cows in the coastal districts of South Africa and Queensland, none of which are housed, may all show a fairly high incidence of tuberculosis.

The essential thing is that there can be and shall be continuous close-cropping in a sunny climate, whereby at most times in daylight the ground is wholly exposed to direct sunlight.

Close cropping is not always feasible in tropical or sub-tropical areas, where the rainfall is high, or in cold boggy areas, such as are found in parts of the British Isles. It is most practicable where there is a light, well-drained soil, with average or light rainfall and a mild or warm climate, whereby growth is continuous. On the veld of South Africa, and in many parts of Australia, the conditions are not economically suitable for close cropping, but it is practised nevertheless, thereby creating the necessary conditions for a low incidence and a low virulence of the bovine tubercle bacillus.

The anti-tuberculous value of close cropping is particularly shown in Tasmania itself, where it is only in a few swampy areas, where close cropping is not feasible, that occasional pockets of relatively high bovine tuberculosis incidence are found. The writer's own district is free from such areas: hence the almost complete absence of bovine tuberculosis.

A factor strongly in Queensland's favour, from an anti-tuberculous point of view, is the far more hygienic housing, as compared with Tasmania.

The houses in Queensland, both in town and country, are almost without exception relatively spacious, detached and airy. Generally they are built on stilts as a protection against the ant, with the result that, not only is the house wholly surrounded by fresh air, but much finds its way up through the floors into the rooms.

On the contrary, in Tasmania, both in town and country, the British cottage or industrial slum type of home is all too common, and it will be a long time before a radical change to the Queensland type of house can be made, though sadly overdue from the point of view of this article.

Experience in South Africa

The position in South Africa as regards the relation of bovine to the human tuberculosis is much the same as in Queensland.

In the Orange Free State, where cropping is close, sunshine high, and humidity low, tuberculosis of cows is very rare.

Dr. Noel Viljoen, Veterinary Superintendent of Abattoir at Bloemfontein, informed the writer that on an average only about four cases of tuberculosis were found among 4,000 bovine beasts slaughtered there annually. The Medical Officer of Health at Bloemfontein stated that he knew of no case of human tuberculosis of bovine origin in the Orange Free State.

On the contrary, in Natal, on the coast, where the rainfall is relatively high and close cropping is not always feasible, the incidence of tuberculosis in cows may be as high as 30 per cent.

Nevertheless, neither in Natal nor elsewhere in South Africa is there any evidence to show that tuberculosis of bovine origin is a menace to human beings.

XUM

Dr. Basil F. Sampson, in a letter to the *British Medical Journal* (June 5, 1943), states that only three cases of human tuberculosis of bovine origin have been confirmed in South Africa in the last ten years, in spite of diligent search. This figure is about the same as that in Queensland medical experience.

Experience in the United States

There has been much propaganda from the United States of recent years to the effect that the gigantic veterinary campaign for the eradication of bovine tuberculosis has had a lot to do with the decline in the incidence of human tuberculosis in general and non-pulmonary tuberculosis in particular.

While admitting that there may be some truth in this claim as regards some of the North-Eastern States, where the climate compels, at times, the winter housing of cows, this writer is absolutely sceptical as regards the claim in general.

On the whole, the sunshine ratio in the United States is high, cropping is close, and the humidity is low, and these physical conditions are precisely those which are not conducive to human infection of a serious sort from bovine sources.

It has been shown that in Queensland, without pasteurisation or eradication of tuberculous cows, the death-rate from all forms of human tuberculosis fell in thirty years from one of the highest to the lowest in the world.

The death-rate from non-pulmonary human tuberculosis in Queensland in 1940 was 2 per 100,000, as compared with a death-rate of 4 per 100,000 in the United States.

Consequently the writer feels under no sort of logical compulsion to believe that either the pasteurisation of milk or the eradication of tuberculous cows played a material part in the human statistical improvement in the United States. On the contrary, it may well be that the American medical statistics would have made a better showing, and might even have equalled those of Queensland, if the cows and their milk had been left alone to play the same immunising part they have played in Queensland and South Africa.

The comparison verges upon the comical when it happens, as at present, that members of the American armed forces in Queensland are forbidden to use unsterilised milk because of the danger—among other perils—of tuberculous infection of bovine origin.

Trespassing on Medical Ground

In conclusion, the writer, though not a doctor, would nevertheless dare, because of the faith engendered in him by a research which may be unique in geographical experience, and even in statistical variety, to formulate what he believes to be the essential conditions as a rule for serious human infection with tuberculosis of bovine origin. Here then are the conditions:

1. There must be a high local potential incidence of bovine tuberculosis showing, by a tendency to generalisation, a high virulence of the bovine bacillus. The word potential is inserted advisedly, because even where, by artificial veterinary means, the actual incidence of grossly infected cows is kept low, cases that may escape detection may have the same high virulence and, consequently, be dangerous to human beings.

2. The human subject of bovine infection must, as a rule, be living in a

XUM

relatively sunless humid environment favourable to mass-infection, such as is found where there is overcrowding and under-feeding in dark, ill-ventilated houses.

Where these conditions do not obtain, that is where there is lots of sunshine, close cropping, plenty of animal food, no overcrowding, and a bovine tuberculosis either rare or of low virulence, as shown by a small tendency to generalisation and by " non-visible lesions " at autopsy, then neither the cow nor its milk need be feared. On the contrary, both—so far as tuberculosis is concerned—should be left alone to allow the human community to enjoy the advantages of some degree of immunisation without running any corresponding risk of infection.

And here the writer leaves it to prosperity to determine the exact or approximate border line, on one side of which bovine tuberculosis is a blessing and the other a curse to mankind.

Australia, South Africa, and probably a large part of Southern Europe, as well as most of United States territory, are definitely on the " blessing " side of the line. The writer believes that applies to much of Devon and Cornwall, and certainly to the Channel Islands.

Postscript for the Unorthodox

Confirmed pasteurisers and incurable subjects of lactophobia should avoid this final section. It is possible that the " border line " postulated in the preceding section will be found by investigators in the light of the future statistical experience to have no existence, because it may transpire that, on balance, bovine tuberculosis is nowhere a curse but, again on balance, everywhere a blessing to humanity.

Those areas—such as Northern England and Southern Scotland—where the physical-chemical factors are most favourable to a high incidence of bovine tuberculosis and the tendency to generalisation of the bovine disease is greatest, may prove, statistically, to be also those areas where humanity stands most in need of whatever immunising value there may be in raw cow's milk. It will not be easy, and—for statistical reasons—must take a long time, to determine the point. One great predominating factor—that of a non-replacement birth-rate and an ever-rising age level of people—cloaks the influence of all other factors, including medical measures, which at the present time are affecting the statistics of human tuberculosis in most civilised countries.

Apart from the war and other man-made calamities, nothing now can wholly stop the decline in the incidence of human tuberculosis so long as an ever-increasing proportion of the population is continually passing out of the tuberculosis-age zone into the cancer-age zone.

Thus if raw cow's milk in Britain, for instance, really has an immunising value for the human being, the effect of general pasteurisation and eradication of tuberculous cows at best would only cause a slowing down of a progressive decline in the mortality from human tuberculosis which must automatically result—apart from a rising age level—from smaller families with consequent less crowding, better nutrition, and less mass-infection.

In time, however, the truth will appear in Britain and elsewhere, as there are grounds for supposing that it may already have appeared in Tasmania, and only time will show whether it is the same truth.

XUM

CHEMICAL CAPONS

By A. H. GUINN,
Kansas City, Missourie

MUCH interest has been aroused among American veterinarians and poultrymen by the work of Lorenz at the University of California, who has reported the "chemical castration" of young roosters after sub-cutaneous implantation of diethylstilbœstrol pellets.

Male birds ranging in age from eight to ten weeks were used in the experiments. A highly-compressed pellet containing an average 15 milligrams of stilbœstrol is implanted by making a one-fourth inch incision in the skin of the neck, then pushing the pellet about one inch forward from the point of incision.

Treated birds gradually lost all male sex characteristics and, to all external appearances and by critical tests of tissues, become true capons. No return of male sex characteristics was apparent when the treated birds had reached a weight of five or six pounds. Old roosters receiving stilbœstrol implants show a great improvement in body fat and meat quality.

It is very important that such pellet implants be made in the discarded or unedible portions of the bird—as the upper cervical region—for consumption of tissue containing either a whole or residual stilbœstrol pellet might cause illness in those who consume such birds.

A GENERAL PRACTITIONER'S METHOD OF MASTITIS CONTROL

By F. M. WILLIAMSON, D.V.M.,
Bluffton, Indiana

BOVINE mastitis has long been my pet subject, a sort of specialty in general practice. Largely for my own interest and enjoyment, I have tabulated records of tests and treatments. At the present time I have more applications from herds than can possibly be handled and still maintain a general practice.

To date, 41 separate herds, totalling 779 cows, have been tested. This adds up to 3,116 quarters, and of these 623, or approximately one out of every five, quarters were infected. This degree of infection has varied from 152 infected quarters out of 184 in one herd, to the lowest which had seven infected quarters out of 128.

My method of testing is as follows: Milk samples are collected individually from each quarter in a sterile tube containing 0.5 c.c. Hotis solution, drawing 9.5 c.c. of milk as aseptically as is possible. We usually try to get to the farm just before milking, as it seems a higher percentage of reactors may be picked up at that time. Samples are incubated for 24 hours in a home-made incubator, using an electric light bulb as a source of heat. This is also equipped with a thermostat, purchased from a mail order house, which varies less than two degrees in accuracy. At the end of 24 hours the samples are inspected and all showing reactions are removed after noting them on a chart. The remaining negative tubes are incubated for another 12 hours and we always find a few

XUM

more positive tubes at that time. This margin of extra incubation has largely removed any percentage of error when compared to microscopic examination of the milk samples. In fact, a microscopic examination is not used at all at the present time.

On the return call we try to arrive shortly after milking, so that all udders may be palpated and intramammary injections given. All cows which reveal any induration of the udder are condemned and must either be removed from the premises or isolated from the milking line. This is clearly understood by the owner before I will even consent to check his herd. All remaining positive cows with soft, pliable udders are treated by injecting the infected quarters with 60 c.c. of an aqueous solution of gramicidin and tyrothricin, such as Ty-Sin. This is a larger dose than is usually recommended, but I find it increases the efficiency of the treatment considerably. The solution is milked out at the following milking period.

Within ten days to two weeks we return and again check the herd. All quarters that have not cleared up are re-treated and another test is run on these quarters following this injection. We plan to keep this up until the quarters become free from all infection. Our own experience is that we have had more success with the aqueous solutions of tyrothricin than with those carrying an oil base.

Of the 623 infected quarters found, 518 were treated, while the remaining cows with infected quarters were condemned. Of the 518 quarters treated, only 11 failed to clear up under repeated treatments. Most quarters are clear following one injection, a few require two, and fewer still require three. We have never had any quarters require more than three treatments except the 11 which failed to respond at all.

The treatment produced undue swelling and decrease of milk flow in two quarters only of the 518 treated. Dairy Association records show that production was increased considerably in the treated animals.

We use either a gravity flow from a Simplex intravenous outfit or a Shikles syringe in making the injection; either method is satisfactory. If the Shikles outfit is used, the injections must be made slowly.

It might also be noted that udders containing thick, ropy milk, with inflamed quarters, are not injected until treated symptomatically and the swelling reduced.

Abstracts of Current Literature

IMMUNITY TO VIRUS DISEASES*

CONCEPTIONS concerning immunity to virus diseases have required constant modification as new knowledge of virus activity has been brought to light, particularly in respect to the nature of their antigenicity, duration and solidity and the immunity produced or its non-existence. Confusion has arisen from the absence of cross-immunity between types of virus (influenza, African horse sickness, foot-and-mouth disease), the disappearance of viruses from the body without leaving protective antibodies behind, and the sojourn of viruses in the

* *The Yale Journal of Biology and Medicine*, May, 1944.

XUM

body in latent form until lowered resistance revives their activity. The diversity of portals of entrance, and kind of tissues they attack, the systemic injury they produce, and the local phenomena (exudation, proliferation, necrosis) which they excite combine to make virus pathology controversial. Among the viruses, there is similarity only in respect to their intracellular delectation—their universality of habitat—and, to a considerable extent, in regard to their tissue specificity. In foot-and-mouth disease and vaccinia, for example, the deeper tissues defend themselves successfully against the virus-raided membranes. Virus inoculated into muscle tissue does not find its way into the susceptible tissues but is pathogenic when injected into the latter.

Another unmeasurable character of viruses is their homogeneity. Though similar in clinical and epidemic terms, there are sharp immunologic differentiations to be made in a number of virus diseases almost pathologically identical and caused by the same basic agent but of different types. Eastern and western and Venezuelan equine encephalitides, St. Louis, West Nile and Japanese B, although encephalitis, etiologically and clinically alike, react to different serologic tests and thereby account for repeated attacks of the same clinical disease as is now known to be the case in human influenza. In fact, the study of virus disease immunity is now carried out with an awareness that immunologic types must be reckoned with primarily to clarify the question of duration of immunity against generalised virus infections which are practically identical but immunologically unlike.

PHENOTHIAZINE-SALT MIXTURE, 1 TO 14, IN CONTROLLING NEMATODE PARASITES IN SHEEP*

THIS controlled observation, involving 1,000 ewes and lambs, was conducted in Southern Illinois, a region where internal ovine parasitism is a constant and pressing problem. All ewes were treated with 1 oz. (28.34 gm.) of phenothiazine just before they were placed on this experiment in January. One-third of the ewes, together with their lambs (lot 1), were used as controls and also as a source of infestation for sheep being fed a phenothiazine-salt mixture. The remaining ewes were divided into two equal groups (lots 2 and 3), both of which were fed 1 : 14 phenothiazine-salt mixture, free choice, throughout the entire pasture season. After the first month at pasture, the sheep in lot 2 were placed upon the pasture recently occupied by the parasitised sheep in lot 1. At the end of the second month at pasture, the sheep in lot 2 were badly parasitised and had sustained severe damage. The sheep in lot 3 were not at any time during the summer placed upon pasture occupied during the current grazing season by parasitised, untreated sheep. None of the sheep in lot 3 developed clinical manifestations of parasitism, although most of them did harbour some worms. They showed by far the highest average gain in weight and the lowest mortality.

In summary, a 1 : 14 phenothiazine-salt mixture given free choice throughout the grazing season protected ewes and lambs against clinical parasitism when the sheep were relatively free from internal parasites at the beginning of the trial and were not at any time allowed access to pasture recently contaminated

* *American Journal of Veterinary Research*, July, 1944.

by parasitised, untreated sheep. The same phenothiazine-salt mixture was not, on the other hand, able to protect similar sheep when they were placed in pasture heavily contaminated with viable parasitic ova.

LIVER THE SOURCE OF ACETONEMIA OF DAIRY COWS*

Two independent researches have traced the cause of acetonemia of dairy cows to faulty hepatic function. The one by Shaw (*J. Dairy Science,* December, 1943) attributed acetonemia to low reserve of hepatic glycogen; the other by Patton (*Veterinary Medicine,* July, 1944) incriminated vitamin A deficiency which the liver normally prevents if the intake of that factor is sufficient. Shaw found liver damage or lazy liver responsible for the recurrence of the disease year after year, and Patton demonstrated vitamin A deficiency by extensive studies in Mississippi where acetonemia is common in the spring after a long winter of vitamin A privation (dry feeding), disappears largely when the cows are turned out to pasture. Patton reports the curing of acetonemia in Mississippi cows, like magic, by injections of vitamin A and the ability to prevent attacks with the same treatment. The latter investigator asserts that many southern cattle (in winter) are borderline cases of acetonemia. In view of the increasing incidence of this catabolic aberration of dairy cows, especially in the southern border states, these findings are regarded as extremely significant since they point out the preventive and the curative treatment.

* *Journal of Dairy Science,* December, 1943; *Veterinary Medicine,* July, 1944; *Journal of the American Veterinary Medical Association,* August, 1944.

THE RELATION OF MINERAL METABOLISM TO ANIMAL HEALTH†

THE amount and kind of mineral elements required to maintain the health of farm animals are as imperative as is adequacy of proteins, carbohydrates and vitamins. It is, therefore, necessary to know what mineral ingredients animal feeds contain in order that proper supplements may be added to rations derived from mineral-depleted soil. In addition to the main mineral elements of which the body ash is composed (calcium, phosphorus, sodium, potassium, sulphur), account must be taken of the trace minerals (iodine, copper, zinc, cobalt, manganese, *et al.*), since all of these have a role to play in the metabolic processes of the normal body. To approach more nearly the level of an exact science, the mineral elements believed to be essential for animal health are checked against the intake of them in the rations. While exactitude in this respect is far from complete, general measures for remineralising both animals and soil have great value for all classes of livestock. Minerals are needed most in growing, pregnant and lactating animals, calcium and phosphorus to provide a sturdy skeleton; chlorides of sodium, potassium and calcium to maintain osmotic pressure; iron, copper and possibly cobalt to sustain hæmoglobin; and iodine is essential to the secretion of thyroxin.

The diseases prevented by the remineralisation of soil and animals are

† *Animal Pathology Exchange,* July, August, September, 1944, with editorial interpolations.

XUM

osteomalacia, rickets, pig anæmia, convulsions in the young, porosis (slipped tendon) in fowls, goiter in calves, grass tetany, and hairless newborn mammals and featherless chickens, all of which are independent of, or associated with, avitaminoses. Ground limestone, defluorinated phosphate rock and bone meal, to which trace minerals are added in proportions yet to be determined through physiologic studies and chemical analyses, transcribe the ambit of successful feeding.

Correspondence

"THE RELATIONSHIP OF BOVINE TO HUMAN TUBERCULOSIS"

To the Editor, THE VETERINARY JOURNAL.

ON the afternoon of the very day my article* was finished, and, in fact, on its way to London, it happened by a trick of fate that a three years' silence from the local abattoir in the matter of bovine tuberculosis was broken.

The voice of the municipal meat inspector on the telephone announced that a case of generalised tuberculosis had been found in a cow slaughtered and now awaiting my inspection at the abattoir.

Fellow missionaries and crusaders will recognise the slight sinking of the heart experienced at my end of the telephone.

Had I been wrong, then, all along, and was this the herald of a great wave of bovine tuberculosis in my part of Tasmania which would nullify and mock all that I had written?

Had I grossly deceived the trusting readers of THE VETERINARY JOURNAL, as well as myself, by publishing a mistaken impression?

Generalised tuberculosis—what memories it awakens! That, for instance, of the condemned cell at the Edinburgh abattoir, which once or twice a week we visited under the tutelage of Stockman, Whitecross, or even red-bearded Tam Dewar himself—dear, dim visions of the academic past.

Those great, grisly harvests of "grapes"; those cavernous abscesses; that endless series of pleura and peritoneum so richly festooned with disease; that almost beautiful mass of lung riddled with miliary tubercle that rolled like shot between the fingers!

"This is it," said the Tasmanian meat inspector, indicating a lot of offal on a slab. "See here," pointing to a small lesion in the liver about the size of a child's marble, and a smaller speck or two in the same organ.

"Yes," one murmured, "it might be—probably is—T.B."

"And here," added the inspector, exhibiting a mediastinal lymphatic gland with a lesion, also suggestive of tuberculosis.

"And look at this retropharyngeal gland," indicating a minute and doubtful abnormality, indeed.

"Well," I said, with my mind still back in Scotland, "now let us look at the carcass," and was led to as fine a body of apparently cow-beef as could be found in all Australia. No evidence of disease in any of the body-glands.

"You are passing this, I suppose," said I, thinking of the beef passed for

* Published in this issue.—Ed.

human consumption in my youth, with Scottish economy, hardihood and—as I think—common sense, after condemnation of fore or hind quarters, most horribly diseased.

"Certainly not," replied the inspector grimly, and clearly rather shocked by my levity. "It is a case of generalised T.B., with lesions both before and behind the diaphragm, and it will be wholly condemned."

"Of course," I apologised, "my mind was elsewhere when I asked the foolish question."

Here, then, is the contrast I have tried to draw in my article—the contrast between bovine tuberculosis that really *is* tuberculosis, achieved by a lusty germ in its optimum environment, and the poor little imitation we have of it, for the most part, in these sunny, close-cropped lands.

Here, in graphic form, is a typical instance of that contrast, and here is why I have come to wonder and to write about the local fear of a danger which, in comparison with that, say, of Scotland, is hardly more real than is the risk of being killed by a star falling from the Southern Cross.

Yours faithfully,

J. W. RAINEY.

APPOINTMENT

MR. JOSEPH EDWARDS, M.Sc., has been appointed by the Milk Marketing Board to be head of the Board's Dairy Husbandry Department.

It is a new department, and he will be responsible for its development. Amongst other duties, he will take charge of the artificial insemination centres to be operated by the Board. Mr. Edwards was engaged formerly at the School of Agriculture at Cambridge, and at present he is serving with U.N.R.R.A.

We offer our congratulations to Mr. Edwards and wish him success in his new post.

Publishers' Notices

Copy of advertisements should be in the hands of the publishers—Baillière, Tindall and Cox—not later than the 1st of the month, if proof is required.

Letters for the Journal, literary contributions, reports, notices, books for review, exchanges, new instruments or materials, and all matter for publication (except advertisements) should be addressed to the Editor.

Binding Cases for Volume 99 and any previous volumes can be obtained from the Publishers. Price 3s. 9d. post free.

Annual Subscription, 25s. ($6.00 U.S.A. currency, post free.)